degenerative changes in heart, kidneys and walls of the blood vessels, of which Doctor Legge speaks, lowering efficiency and resistance and undoubtedly causing a form of chronic invalidism which may be one of the basic causes for the high death rate from heart and kidney diseases among the workers in these places.

Carbon monoxid is a serious factor in the housing of people in large cities. Cooking, eating and sleeping in one room with unventilated gas stoves has been the cause of thirty-one deaths out of one hundred and eighteen credited to carbon monoxid poisoning in San Francisco from January to December of 1927. Eighty-seven were attributed to suicide, but again no positive evidence exists that any of these deaths were suicide. The defective rubber tubing connecting gas plates and gas jets, as well as the lack of proper outlets to flues or the open air from gas stoves, cannot be wholly guarded against.

It behooves every health officer to enforce rigidly the housing laws and compel those erecting apartments and transforming the old residences into one and two-room places for housing of families, to see that every kitchen is supplied with a flue to carry off the product of combustion from the gas stoves and to eliminate altogether the gas plate with rubber tube connection.

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ALEX M. LESEM, M. D. (739 Fourth Street, San Diego).—In discussing this paper I wish to state that during the year 1925 our local community experienced a number of cases of carbon monoxid poisoning which were classified as distinctly separate from suicidal attempts.

I believe that the American Gas Association through experimentation in its laboratory has established the fact that carbon monoxid poisoning caused by gas appliances burning natural gas, artificial gas, or mixed gas is due to incomplete combustion. Gas appliances which are designed to burn natural gas do not satisfactorily burn artificial or mixed gases, so a good deal depends upon the intelligent installation of the proper gas appliances.

During the first six months of 1925 forty-three cases of carbon monoxid poisoning which resulted in death were reported to the State Board of Health. Many cases resulted from moving appliances from localities where natural gas was burned to other cities where artificial gas was employed. Another factor proved to be the purchase of worn-out appliances from second-hand stores, together with faulty installation and without municipal inspection.

The third factor is the installation of gas appliances which have never been inspected or approved by the American Gas Association; which are sold for much less money than approved appliances; and which are taken home and installed by the purchaser without proper ventilation.

I wish to limit my discussion to gas appliances responsible for incomplete combustion. I believe that the solution for this particular cause of accidents lies in the education of the community along the lines of proper use and installation of gas appliances.

I believe that legislative bodies in every community should assume responsibility for prevention of such accidents and should require that all piping and gas appliances be installed under the most rigid and competent inspection.

I believe that all physicians interested in the preventive phase of carbon monoxid poisoning should be highly in favor of a state-wide legislation which would educate the consumer and prevent haphazard installations of these appliances which have proved so dangerous to human lives.

SOME OBSERVATIONS ON THE ANEMIAS*

By ROBERT POLLOCK, M. D.
San Diego

DISCUSSION *by Rachel L. Ash, M. D., San Francisco; J. M. McCullough, M. D., Crockett; Willard J. Stone, M. D., Pasadena.*

THAT anemia of the severer grades is seemingly on the increase in this country, is evidenced by the increase of activity in combating it. Official vital statistics do not accurately record the deaths from anemia, partly because the disease with which the anemia is associated usually gets the credit for the death, and partly because of the unfortunate nomenclature in the anemias.

Any marked deviation from normal blood is sooner or later registered on all the functions and tissues of the body; hence anemia of marked degree is always of vital interest to the clinician. The vast amount of research done on anemia during the past year or two warrants a critical analysis of this material. I am attempting to bring forward only such points as may be termed controversial in order by discussion to help toward a better understanding. I find here a subject wherein etiology, diagnosis, treatment and prognosis all furnish abundant material for speculation and diversity of opinion. However, scientific truth is an ever changing, constantly shifting entity, and the last word has never been said on any subject.

CLASSIFICATION

An adequate working classification of the anemias is the following:

1. Anemias due to hemorrhage: ulcer, hemorrhoids, nephritis, urologic conditions, cancer and casualty.
2. Anemias due to hemolysis from toxins, known or unknown, such as malaria, sepsis, chemical bodies, hemolytic jaundice, some cases of pernicious anemia.
3. Anemias due to disease or defective function of the blood making tissues, such as the aplastic anemias, some cases of pernicious anemia.

So-called pernicious anemia is probably most often due to hemolysis from unknown toxins, although some of these cases are probably true aplastic anemias. The aplastic cases are as a rule rapidly fatal and occur before middle age.

Usually when hemolysis is taking place we have an increase of the urobilinogen in the urine and a yellowing of the skin.

Most of the discussion of the past year has centered about the so-called pernicious anemia, chiefly on account of the renewal of interest in its dietetic treatment. Here is an anemia showing a marked degree of hemolysis, accompanied by evidence of increased regeneration of cells, such as the appearance of nucleated red cells and megaloblasts. The red cells show many irregularities in form with a comparative increase in their size over those in other anemias as evidenced by their cell volume. This is accountable for the high color index in this anemia. To rely upon the color index in the matter of differential diagnosis between a

* Read before the Southern California Medical Association meeting at Los Angeles, November 25-26, 1927.

primary and secondary anemia seems futile. This division into primary and secondary anemias I look upon as of purely academic interest, and of no practical purpose.

The many appliances used to determine hemoglobin percentage depending upon the eyes' ability to match color tones, the variations in technique in making blood counts, all tend to minimize the value of the color index in borderline cases. The general condition of the blood can best be told by a close study of individual cells as to size, shape and other abnormalities; and the differential diagnosis between the anemias should be determined largely upon the clinical symptoms, the physical examination and the history.

Since the passing of chlorosis, which is rarely discussed in the literature of today, pernicious anemia is the only so-called primary anemia demanding discussion and since we admit that we do not know the cause of it, is not the use of the word "primary" rather an arbitrary distinction suggestive of our ignorance? Certainly in every case of anemia we should search exhaustively to find a cause before labeling it a primary anemia.

Let us admit that disease, like life itself, does not exist *de novo,* but has a causative factor which it is the function of science to bring to light. Patients with severe anemia have quietly expired while we were still debating the question whether their anemia was primary or secondary. I feel that medical nomenclature could well dispense with such words as primary, essential and idiopathic when used as a cloak for our ignorance; the chief purpose served by such an adjective as "pernicious" is to strike terror into the layman's heart.

In our classification of the anemias, pernicious anemia falls in line as an anemia due to hemolysis from as yet unknown toxins. The present tendency is also to attribute to the disease a definite hereditary tendency. Undoubtedly some of the unmanageable cases of "pernicious anemia" that do not respond well to any treatment are cases of true aplastic anemia.

The unknown toxins in pernicious anemia are being assiduously sought in connection with bacterial strains and protozoal and other parasites of the intestinal tract; and it is here that a certain similarity between sprue and pernicious anemia has been dwelt upon. It would seem to me, however, that the rather characteristic gastro-intestinal picture in sprue ought not to be confounded with the symptoms accompanying the achlorhydria of anemia. Neither the achylia nor any other of the outstanding associations of pernicious anemia seem to account for the anemia. Both the achylia and the degenerative changes in the spinal cord at times occur before the typical blood picture of advanced anemia is present; but these are probably caused by certain toxins at work in the system rather than being either the cause or the result of the anemia.

At all events we are in no position to accept the final word on this point. It is of interest that among the most outstanding pathologic changes found after death from pernicious anemia, are these degenerative changes in the nervous system. Similar changes, however, are also found in diabetes, pellagra, leukemia and other diseases.

Dr. Robert Preble, whom I consider a high authority and excellent teacher, speaking of pernicious anemia, says "that the anemia should be recognized as only one part of a comprehensive entity in which the digestive system, the nervous system, and the endocrine system are also involved."

TREATMENT (AFTER WALES, BARKER AND OTHERS)

1. Preventive.
   - (a) Heredity.
   - (b) Early diagnosis.
   - (c) Oral and general hygiene.
   - (d) Early removal of known foci of infection or source of toxemia.
2. Symptomatic.
   - A. To decrease hemolysis.
     - (a) By radiation.
     - (b) By splenectomy.
   - B. To supply new blood and to promote regeneration of blood.
     - (a) By transfusion.
     - (b) By drugs.
     - (c) By diet.

PREVENTIVE TREATMENT

Preventive treatment always appeals to me, and I like to think of an *early diagnosis* as a part of preventive treatment, although it sounds a bit paradoxical. Nothing is surer, however than that the detection of an anemia when the blood is only 20 to 25 per cent below normal gives the physician a great advantage, and I believe that all anemias of this grade call for careful treatment.

Many conditions which lead to severe anemia should arouse our suspicion by such symptoms as dyspnea, weakness, numbness of the extremities and gastro-intestinal complaints long before marked changes in the blood picture have appeared.

The question of *heredity* is being given more attention recently; although we can do little with it in the way of treatment except to make careful blood examinations. The annual or semi-annual physical check-up so broadly advocated today should include a blood count and usually a blood Wassermann.

Oral and General Hygiene.—The dentist checks more carefully upon the prevention of trouble in the mouth than physicians do with the rest of the body. General hygiene is being splendidly taught in our public schools today.

Early *removal* of all *foci of infection* appeals to most of us, especially those of the surgical persuasion. Just at present the gall bladder seems to be bearing the brunt of the attack, while the intestine with its twenty feet of mystery is suspected of originating many toxins. Intensive study of intestinal protozoa has been stimulated because of their known tendency to produce hemorrhages and achlorhydria.

SYMPTOMATIC TREATMENT

The agents used *to decrease hemolysis* seem to me to be of questionable value in arresting severe anemias. Radiation has proven of some value in

splenic anemia, and splenectomy of no permanent value in any anemia.

To supply new blood and *produce regeneration* of the blood I think that we have in repeated blood transfusion, with small amounts of carefully matched blood, a remedy of unquestionable value.

Drugs play rather a secondary part in the treatment of anemias, although arsenic intelligently used will often stimulate the blood-forming tissues. Where achlorhydria is present the use of HCl is indicated, and it is given usually in much larger doses in anemia than in other achylous conditions.

At present the treatment by dietetic measures is being powerfully stimulated. Ever since Minot and Murphy published their first article on special feeding in pernicious anemia, the literature has been teeming with articles on the dietetic treatment of anemia. The revival of the use of gland foods, liver, kidney and pancreas, and the building around them a definite system of diet made up chiefly of these organs in liberal amounts—200 to 300 grams daily with the free use of muscle meats and large amounts of fresh vegetables and fruits—has produced such strikingly good results that it has suddenly become the accepted treatment for all severe anemias. Spleen and marrow substances are also used as in the past, but at present liver is easily the favorite, and we can well afford to express our resourcefulness in devising attractive ways of serving it to the none too vigorous appetites of our anemic patients.

Just what principle in liver is so efficacious has not as yet been brought out, and the theory that vitamin A deficiency in the diet, for a long time continued, will cause severe anemia is not as yet definitely proven, and Doctor Minot himself does not favor it. However, for the time being the dietetic treatment has the call in the treatment of anemia and we find transfusion at times an invaluable aid.

In conclusion let us think of anemia as a grave condition that should be diagnosed early and carefully watched. Let us think of all anemias as probably secondary to some condition which it is our duty to uncover and correct. Let us try to feel that no anemia no matter how labeled, is necessarily beyond treatment, and while applying such therapeutic measures as we possess, let us continue to search assiduously for an underlying cause. As special diets are proving to be of marked value in their treatment, it is reasonable to suspect that some dietary deficiency, possibly a vitamin, may be a causative factor in the production of some anemias. As diet has not as yet restored the stomach's chemistry the free use of HCl should still be continued. Outside of diet and HCl, arsenic and intelligently applied transfusion are our best helps.

LATER STATISTICS

Since the above was written, through the courtesy of Dr. W. P. Shepard of the Metropolitan Life Insurance Company's office in San Francisco, I have secured the following statistics taken from the twenty-fifth annual report of the Federal Bureau of the Census:

Total deaths from anemia in 1923 were 5867 or 6.0 per 100,000 population; in 1924 were 6167 or 6.2 per 100,000 population.

Total deaths from pernicious anemia in 1923 were 5169 or 5.3 per 100,000 population; in 1924 were 5478 or 5.5 per 100,000 population.

Total deaths from anemia other than pernicious anemia in 1923 were 708 or 0.7 per 100,000 population; in 1924 were 689 or 0.7 per 100,000 population.

TABLE 1.—*Deaths from Anemia and Per Cent Per 100,000 Poulation*

	Pernicious Anemia	Anemia Other Than Pernicious	Total Deaths
1923	5159 or 5.3%	708 or 0.7%	5867 or 6%
1924	5478 or 5.5%	689 or 0.7%	6167 or 6.2%

1301 Medico-Dental Building.

DISCUSSION

RACHEL L. ASH, M. D. (490 Post Street, San Francisco).—Doctor Pollock's review of the severe anemias is both comprehensive and timely. He calls our attention to the necessity of differentiating the various types, offers a practical classification for clinical purposes, and pleads a more universal recognition of pernicious anemia. He insists on the importance of early detection and careful treatment of all low-grade anemias by modern methods. That there is still very little known of the etiological factors, and that the blood picture is by no means the first clinical manifestation of grave anemias, is carefully emphasized by the writer. It is interesting to note in the appended statistical report, that deaths from the grave anemias, including pernicious anemia, seem to be on the increase.

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J. M. MCCULLOUGH, M. D. (Crockett).—Doctor Pollock's paper expresses the most modern views on the anemias. He calls our attention to certain symptoms that indicate a beginning anemia. Certainly these cases should be kept under observation, as the suggestion that the lesser grades of anemia be given close attention is very good. Undoubtedly many of the severe anemias could be prevented by earlier diagnosis and removal of foci of infection.

Minot and Murphy have given us a much more efficient method of treating pernicious anemias, and I believe that by carrying out these measures it will be possible to show a decrease in the mortality rates instead of the apparent increase that the attached statistics now show.

In closing I wish to add that more attention should be paid to carbon monoxid and lead as a factor in many anemias.

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WILLARD J. STONE, M. D. (65 North Madison Avenue, Pasadena).—Doctor Pollock's observations on the anemias cover an interesting field in clinical medicine. I agree with his view that the color index is of little relative value in differentiating so-called primary from secondary anemia. Since the color index is dependent upon the estimation of hemoglobin by color matching and the ability to recognize shades is so variable in the different individuals who are called upon to make the tests, the findings are often inconclusive. It may be emphasized that the hemoglobin of so-called normal men, women and children varies, and that the limits of normal vary in different parts of the country (*i. e.*, at the seashore level, in cold as contrasted with warm semitropical areas, or in altitudes above 4000 feet). Under a given oxygen pressure it is found that less oxygen is taken up by hemoglobin the higher the temperature. It may also be emphasized that the various so-called standards vary. The Dare hemoglobinometer in common use in this country has for its standard a hemoglobin content of 13.77 gm. per 100 cc. for 100 per cent, the Sahli hemoglobinometer

also commonly used has for its standard a hemoglobin content of 17.2 gm. per 100 cc. for 100 per cent, while the Hellige standard is 17.0 gm. per 100 cc. for 100 per cent. Variations in cell volume must affect the determination enormously. In hospital work it has not been uncommon to note a variation of 15 per cent in the readings taken by different interns or technicians. I sympathize therefore with Doctor Pollock's point that until some more accurate method is devised for the clinical determination of hemoglobin its importance is a relative one only, and slight variations are of no significance with present methods. His remark that splenectomy has proven of no permanent value in any anemia I think is open to question. In many cases of splenic anemia great improvement occurs after removal of the spleen. The same may be said to follow removal of the spleen in the anemia of hemorrhagic purpura. Those who are interested in this phase of treatment should read the excellent article on splenectomy by W. J. Mayo in the *American Journal of the Medical Sciences* for March, 1926. In general we may feel more hopeful than a few years ago about the outlook in the severe anemias. The work of Minot and Murphy on liver feeding, and the vitamin deficiency studies of the late Karl Koessler of Chicago, have pointed the way to new investigations which give great promise.

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DOCTOR POLLOCK (closing).—I am grateful for the discussion elicited by my paper, which was written last October (1927), with the avowed intention of "bringing forward such points as may be termed controversial in order by discussion to help toward a better understanding."

Much has been written concerning anemia during the intervening ten months, and, as Doctor Stone well says, "In general we feel more hopeful than a few years ago about the outlook in the severe anemias."

Let us assiduously search for the etiologic factors and study the pathological tissue changes in the various conditions associated with anemia.

THE LURE OF MEDICAL HISTORY

WILLIAM HARVEY*

PART I

The Life of the Author of *De Motu Cordis*

By FRANK H. RODIN, M. D.
San Francisco

"The Heart of creatures is the foundation of life, the Prince of all, the Sun of their Microcosm, on which all vegetation does depend, from whence all vigor and strength does flow."—From the dedication to Charles I.

ON May 14, 1928, delegates from universities and medical societies throughout the world met in London to attend the celebrations arranged by the Royal College of Physicians of London in honor of the three hundredth anniversary of the publication of William Harvey's great work, the *De Motu Cordis*.

Harvey lived during the last quarter of the sixteenth century and the first half of the seventeenth. This was a period of great political and social upheaval in England and on the Continent. In 1603 Queen Elizabeth died. Fifteen years later Sir Walter Raleigh was executed. It was the period of the Long Parliament (1640-1653) and the civil wars in England and Scotland (1642-1652). The Thirty Years' War was waged on the Continent (1618-1648). In 1649 Charles I, to whom Harvey dedicated his work, was executed. The divine right of kings was being questioned and the supremacy of the Catholic Church was being challenged. Slowly the old order was giving way to the new. Francis Bacon wrote his *Novum Organum* (1620) advocating the inductive system of reasoning, which is held by many to have inspired Harvey.

It was still heresy to question the old established beliefs, especially ecclesiastical. Servetus, who was a predecessor of Harvey, gave a correct description of the pulmonary circulation in a theological book entitled *Christianismi Restitutio*, published at Lyons in 1543. The book contained doctrines for which Calvin caused him to be burned with his book.

> John Calvin, whose peculiar fad
> It was to call God murderous;
> Which further led the feverish cad
> To burn alive the Servetus.

In 1600 Giordano Bruno was burned at Rome. Galileo in 1632 was forced to abjure his scientific creed before the Papal tribunal.

The Renaissance in scholarship and letters had been well established. Astronomy was being placed on a new basis, and a new science of physics was beginning to spring up from Harvey's contemporaries, Galileo, René Descartes, and Borelli. About the same time chemistry was being placed on a rational basis by the work of Paracelsus and Van Helmont.

For over fourteen centuries Galen was the undisputed authority of medicine, with only a faint voice here and there such as Vesalius and Servetus, in the sixteenth century, questioning with great temerity this authority. Harvey's discovery of the circulation of the blood freed medicine and natural science from dogmas, superstition, and confusion and placed them upon a rational and scientific basis. This discovery made it possible for subsequent physiologists to work out the problems raised by Harvey, free from the limitations imposed by tradition.

BIOGRAPHICAL SKETCH

William Harvey was born at Folkestone, Kent, on April 1, 1578. The place of his birth was a building of some importance, as later it became a posthouse. Very little is known about the Harvey family prior to William's father, Thomas Harvey, who married Juliana Jenkin, who died a year later, leaving him a daughter. Thomas Harvey soon married again and William was the first child of the second union. The father was a man of some importance, as he was a jurat or alderman of Folkestone and became mayor of that city in 1600. Very little is known of William's mother. There were seven sons and two daughters in the family. The second son held many important positions with the court and government, and was also a member of parliament for Hythe. The remaining five sons were very successful in business and became great merchants.

William's early education was obtained at Folkestone. When ten years of age he attended the grammar school at Canterbury. When sixteen

* Part I deals with a history of the life of William Harvey, and Part II, which will appear in the January issue, with Harvey's great work, De Motu Cordis.